

Letter to the Editor: Alkali Therapy, Hyperphosphatemia, and Acute Tumor Lysis Syndrome

High-dose allopurinol therapy, hydration with hypotonic fluids, and urinary alkalinization are now routine in the management of patients with lymphoid malignancies at high risk for the acute tumor lysis syndrome [1]. The key to the successful management of these patients is the establishment of high flow rates (>250 mL/h) of maximally dilute urine before the institution of chemotherapy. With current management protocols, hyperuricemic nephropathy is rare in the posttreatment period [2–4]. The major metabolic problem following chemotherapy in patients with high-grade lymphoid neoplasms is hyperphosphatemia causing intrarenal precipitation of calcium phosphate [2–4]. The risk of hyperphosphatemic renal failure is increased in the presence of alkaline urine and hence measures to attain alkalinization of urine should be discontinued at the start of chemotherapy [1,3]. This aspect of management does not appear to be widely recognized, and cases of acute tumor lysis with hyperphosphatemic renal failure related to alkali therapy continue to be reported [4–6].

In the patient described by Vachvanichsanong and colleagues [7], alkali administration could have been discontinued at the time of starting of prednisone therapy. Information about the urine flow rates in the days immediately following prednisone therapy would have been helpful in understanding the metabolic aberrations and the timing of hemodialysis in the patient.

The lack of calcium phosphate deposits in the renal biopsy specimen may have been due to the timing of the renal biopsy. It is also possible that the fixative used

(aqueous formalin) had caused the dissolution of the phosphate deposits in the biopsy sample.

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